

10/806288

(FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO, PHIC, PHIN, TOXCENTER, PASCAL, FEDRIP, DISSABS' ENTERED AT 09:14:38 ON 06 OCT 2004)

L1 178 SEA ABB=ON PLU=ON "GUSS B"?/AU - Author(s)
 L2 143 SEA ABB=ON PLU=ON "FRYKBERG L"?/AU
 L3 516 SEA ABB=ON PLU=ON "FLOCK J"?/AU
 L4 1653 SEA ABB=ON PLU=ON "LINDBERG M"?/AU
 L5 2935 SEA ABB=ON PLU=ON "NILSSON M"?/AU
 L6 8 SEA ABB=ON PLU=ON L1 AND L5 AND L2 AND L3 AND L4
 L7 138 SEA ABB=ON PLU=ON L1 AND (L5 OR L2 OR L3 OR L4)
 L8 33 SEA ABB=ON PLU=ON L5 AND (L2 OR L3 OR L4)
 L9 49 SEA ABB=ON PLU=ON L2 AND (L3 OR L4)
 L10 26 SEA ABB=ON PLU=ON L3 AND L4

 L13 28 SEA ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5 OR L7 OR L8 OR L9 OR L10) AND STAPHYLOCOCC?(S) (COAGULASE(W) (NEG OR NEGATIVE))
 L14 33 SEA ABB=ON PLU=ON L6 OR L13
 L15 14 DUP REM L14 (19 DUPLICATES REMOVED)

L15 ANSWER 1 OF 14 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2004:278972 BIOSIS
 DOCUMENT NUMBER: PREV200400283115
 TITLE: Fibrinogen binding protein originating from
coagulase-negative staphylococcus

AUTHOR(S): **Guss, Bengt** [Inventor, Reprint Author];
Nilsson, Martin [Inventor]; **Frykberg, Lars**
 [Inventor]; **Flock, Jan-Ingmar** [Inventor];
Lindberg, Martin [Inventor]

CORPORATE SOURCE: Dag Hammarskjolds Vag 238B, S-756 52 Uppsala, Sweden
 PATENT INFORMATION: US 6733758 May 11, 2004
 SOURCE: Official Gazette of the United States Patent and Trademark
 Office Patents, (May 11 2004) Vol. 1282, No. 2.
<http://www.uspto.gov/web/menu/patdata.html>. e-file.
 ISSN: 0098-1133 (ISSN print).

DOCUMENT TYPE: Patent
 LANGUAGE: English
 ENTRY DATE: Entered STN: 9 Jun 2004
 Last Updated on STN: 9 Jun 2004

AB A new fibrinogen binding protein or polypeptide originating from
coagulase negative staphylococci,
 biotechnological methods for producing the protein or polypeptide having
 fibrinogen binding activity and a recombinant DNA molecule coding for the
 protein (or fragments thereof), and micro-organisms (including viruses)
 containing this recombinant DNA molecule. The present invention further
 comprises the therapeutic and diagnostic use of the protein and/or DNA,
 e.g., a diagnostic kit for determining the presence and/or type of
coagulase negative staphylococci and a vaccine
 composition, comprising the protein or DNA.

L15 ANSWER 2 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
 on STN DUPLICATE 1

ACCESSION NUMBER: 2003176032 EMBASE
 TITLE: Extracellular adherence protein from *Staphylococcus aureus*
 enhances internalization into eukaryotic cells.

Searcher : Shears 571-272-2528

AUTHOR: Hagggar A.; Hussain M.; Lonnie H.; Herrmann M.;
Norrby-Teglund A.; **Flock J.-I.**
CORPORATE SOURCE: J.-I. Flock, Department of Laboratory Medicine, Karolinska
Institutet, Huddinge University Hospital, S-141 86
Huddinge, Sweden. jan-ingmar.flock@labmed.ki.se
SOURCE: Infection and Immunity, (1 May 2003) 71/5 (2310-2317).
Refs: 49
ISSN: 0019-9567 CODEN: INFIBR
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB In this study we have shown that Eap (extracellular adherence protein) plays a role in the internalization process of **Staphylococcus aureus** into eukaryotic cells. Eap is a protein that is mostly extracellularly and to a lesser extent is bound to the bacterial surface as a result of rebinding. Eap is able to bind to several plasma proteins, such as fibronectin, fibrinogen, and prothrombin. It has the capacity to form oligomers and is able to agglutinate *S. aureus*. A mutant strain, Newman mAH12 (eap:: Ery(r)), with a deficient eap gene was used in the present study. We have demonstrated that (i) strain Newman mAH12 could adhere to and become internalized to a higher extent by eukaryotic cells than the isogenic mutant, (ii) strain Newman mAH12 complemented with the eap gene displayed restoration of the internalization level, (iii) externally added Eap enhanced the internalization of laboratory and clinical *S. aureus* strains as well as of *S. carnosus* (a **coagulase-negative** species devoid of proteins important for internalization), and (iv) antibodies against Eap were able to block the internalization process in strain Newman mAH12 and clinical isolates. Eap, with its broad binding capacity and its surface localization, thus seems to contribute to the internalization of *S. aureus* into eukaryotic cells. We therefore propose a novel internalization pathway for *S. aureus* in which Eap plays an enhancing role.

L15 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 2003:294443 CAPLUS
DOCUMENT NUMBER: 139:286963
TITLE: GST-Fbe can recognize β -chains of fibrin(ogen) on
explant materials
AUTHOR(S): Pei, Lei; Arvholm, Ingegerd Lofving; Lonnie, Lena;
Flock, Jan-Ingmar
CORPORATE SOURCE: Division of Clinical Bacteriology, Department of
Microbiology, Pathology and Immunology, Karolinska
Institute, Huddinge University Hospital, Stockholm,
SE-141 86, Swed.
SOURCE: Journal of Chromatography, B: Analytical Technologies
in the Biomedical and Life Sciences (2003), 786(1-2),
319-325
CODEN: JCBAAI; ISSN: 1570-0232
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Staphylococcus epidermidis**, a **coagulase-neg.**
staphylococcus (CoNS), is one of the leading pathogens of
nosocomial infections, particularly associated with foreign body infections.

Adherence of *S. epidermidis* to fibrinogen deposited on the surfaces of implants is important for the development of foreign body infections. A gene (fbe) encoding a fibrinogen-binding protein from *S. epidermidis* (Fbe) was identified by shotgun phage display. A portion of fbe was cloned into a GST-fusion vector. Affinity to glutathione-Sepharose by the GST-tag and affinity to fibrinogen-Sepharose by the Fbe part were applied to purify the recombinant Fbe. The purity and efficacy of the methods used in protein purification was compared. Furthermore, the potential physiologic role of Fbe was studied by the interaction between GST-Fbe and components extracted from explanted materials in vitro.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 3

ACCESSION NUMBER: 1998:363168 CAPLUS
DOCUMENT NUMBER: 129:92648
TITLE: A fibrinogen-binding protein of *Staphylococcus epidermidis*
AUTHOR(S): Nilsson, Martin; Frykberg, Lars; Flock, Jan-Ingmar; Pei, Lei; Lindberg, Martin; Guss, Bengt
CORPORATE SOURCE: Department of Microbiology, Swedish University of Agricultural Sciences, Uppsala, S-750 07, Swed.
SOURCE: Infection and Immunity (1998), 66(6), 2666-2673
CODEN: INFIBR; ISSN: 0019-9567
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The present study reports on fibrinogen (Fg) binding of *Staphylococcus epidermidis*. Adhesion of different *S. epidermidis* strains to immobilized Fg was found to vary significantly between different strains, and the component responsible was found to be proteinaceous in nature. To further characterize the Fg-binding activity, a shotgun phage display library covering the *S. epidermidis* chromosome was constructed. By affinity selection (panning) against immobilized Fg, a phagemid clone, pSEFG1, was isolated, which harbors an insert with an open reading frame of .apprx.1.7 kilobases. Results from binding and inhibition expts. demonstrated that the insert of pSEFG1 encodes a specific Fg-binding protein. Furthermore, affinity-purified protein encoded by pSEFG1 completely inhibited adhesion of *S. epidermidis* to immobilized Fg. By addnl. cloning and DNA sequence analyses, the complete gene, termed fbe, was found to consist of an open reading frame of 3276 nucleotides encoding a protein, called Fbe, with a deduced mol. mass of .apprx.119 kDa. With a second phage display library made from another clin. isolate of *S. epidermidis*, it was possible to localize the Fg-binding region to a 331-amino-acid-long fragment. PCR anal. showed that the fbe gene was found in 40 of 43 clin. isolates of *S. epidermidis*. The overall organization of Fbe resembles those of other extracellular surface proteins of staphylococci and streptococci. Sequence comparisons with earlier known proteins revealed that this protein is related to an Fg-binding protein of *Staphylococcus aureus* called clumping factor.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 5 OF 14 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on

STN
 ACCESSION NUMBER: 1999:61336 BIOSIS
 DOCUMENT NUMBER: PREV199900061336
 TITLE: Variation of **coagulase-negative staphylococci** in the skin flora of healthy individuals during one year.
 AUTHOR(S): Jung, K. [Reprint author]; Brauner, A.; Kuhn, I.; **Flock, J.-I.**; Mollby, R.
 CORPORATE SOURCE: Dep. Microbiol., Nova Med. Calab, St. Gorans Hosp., SE-112 81 Stockholm, Sweden
 SOURCE: Microbial Ecology in Health and Disease, (July, 1998) Vol. 10, No. 2, pp. 85-90. print.
 ISSN: 0891-060X.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 16 Feb 1999
 Last Updated on STN: 16 Feb 1999

AB In order to investigate the variation and turnover time of **coagulase-negative staphylococci** (CNS) in the skin flora and whether CNS strains were transmitted over the body, we studied CNS in the skin flora of seven healthy persons during a period of one year. From 134 samples, collected approximately monthly, 547 CNS were isolated. All isolates were typed by PhP-CS, a biochemical fingerprinting method and, if further discrimination was required, also by pulsed-field gel electrophoresis of DNA. We found 109 different strains out twice. The CNS flora of the toes showed a high variation compared to the nares and axillas. Eighty percent of the resident strains colonized the skin for more than three months and two strains were isolated during the whole study period. Different strains were strain is very uncommon. It is concluded that although many different strains can be found on a body location, most of them are only found occasionally. However, certain strains may colonize the skin and become permanent members of the normal flora.

L15 ANSWER 6 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 4
 ACCESSION NUMBER: 1998:31332 CAPLUS
 DOCUMENT NUMBER: 128:85161
 TITLE: Cloning and expression of a new fibrinogen-binding protein gene fig originating from **coagulase-negative Staphylococcus**
 INVENTOR(S): **Guss, Bengt; Nilsson, Martin; Frykberg, Lars; Flock, Jan-Ingmar; Lindberg, Martin**
 PATENT ASSIGNEE(S): Guss, Bengt, Swed.; Nilsson, Martin; Frykberg, Lars; Flock, Jan-Ingmar; Lindberg, Martin
 SOURCE: PCT Int. Appl., 45 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9748727	A1	19971224	WO 1997-SE1091	19970618
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,				

DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9732825 A1 19980107 AU 1997-32825 19970618
 EP 922056 A1 19990616 EP 1997-928617 19970618

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

JP 2001503609 T2 20010321 JP 1998-502819 19970618
 US 6733758 B1 20040511 US 1999-147405 19990401

PRIORITY APPLN. INFO.: SE 1996-2496 A 19960620
 WO 1997-SE1091 W 19970618

AB A new fibrinogen-binding protein, denoted FIG, or polypeptide originating from **coagulase-neg. staphylococci**, biotechnol. methods for producing said protein or synthetic polypeptides having fibrinogen-binding or fibrinogen-inhibiting activity and a recombinant DNA mol. coding for said protein (or fragments thereof), and microorganisms (including viruses) containing this recombinant DNA mol. are claimed. Suitable staphylococci strains include *Staphylococcus epidermidis* strain HB. Plasmid or viral gene vectors are also used to help produce the recombinant fig gene. Host cells include bacterial strains such as *Escherichia coli*, *Staphylococcus* sp., *Lactobacillus*, yeast, and other eukaryotic cells in culture. The gene fig protein may also be present as part of a fusion protein, e.g. with glutathione transferase. The present invention further comprises the therapeutic and diagnostic use of said protein and/or DNA, e.g. a diagnostic kit for determining the presence and/or type of **coagulase-neg. staphylococci** and a vaccine composition, comprising said protein or DNA.

L15 ANSWER 7 OF 14 MEDLINE on STN DUPLICATE 5
 ACCESSION NUMBER: 96085078 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 7488390
 TITLE: Typing of **coagulase-negative staphylococci** from peritonitis in CAPD-patients by the PhP-CS system and REA.
 AUTHOR: Jung K; Brauner A; Kuhn I; Ransjo U; Hylander B; **Flock J I**; Mollby R
 CORPORATE SOURCE: Department of Clinical Bacteriology, Danderyd Hospital, Sweden.
 SOURCE: APMIS : acta pathologica, microbiologica, et immunologica Scandinavica, (1995 Sep) 103 (9) 679-85.
 Journal code: 8803400. ISSN: 0903-4641.
 PUB. COUNTRY: Denmark
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199601
 ENTRY DATE: Entered STN: 19960125
 Last Updated on STN: 19960125
 Entered Medline: 19960104

AB **Coagulase-negative staphylococci** (CNS) were

the most common bacteria causing peritonitis in patients treated with continuous ambulatory peritoneal dialysis (CAPD). In order to investigate if the same clone was responsible for the peritonitis in the different patients and if the exit site was the source of infection we followed 68 patients on CAPD for 2 years. During this period 9 patients had 12 episodes of peritonitis caused by CNS. Cultures were taken from exit site and peritoneal fluid in all patients at peritonitis and during the first study year at monthly intervals. In each culture up to 10 isolates of CNS were randomly collected and frozen. All 437 CNS isolates from the patients with CNS peritonitis were typed using a biochemical typing method and 41 isolates identical by this method were further discriminated by a DNA fingerprinting method. Identical strains were in no case isolated from different patients, indicating that no virulent strain was spread between the patients. The isolates causing the peritonitis were never found at the exit sites before the first day of the peritonitis in any patient. In only two patients was the same strain found at the exit site and in the peritoneal fluid on the first day of peritonitis. It thus seems that no virulent clone of CNS was infecting the patients and we found no evidence of CNS at the exit site causing the peritonitis.

L15 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 6

ACCESSION NUMBER: 1995:474618 CAPLUS

DOCUMENT NUMBER: 122:234508

TITLE: Comparison of epidemiological markers of
coagulase-negative
Staphylococci

AUTHOR(S): Jung, K.; Wilton, J.; Kuhn, I.; Aronsson, B.; Moellby, R.; Flock, J. -I.

CORPORATE SOURCE: Stockholm County Council Microbiological Laboratory, Center Bio-Technology, Novum, Swed.

SOURCE: Zentralblatt fuer Bakteriologie, Supplement (1994), 26, 110-14

CODEN: ZBASE2; ISSN: 0941-018X

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We have compared different methods for typing **coagulase-neg. staphylococci** (CNS) and especially *S.epidermidis*. The discriminatory powers of antibiogram, biotyping, biochem. fingerprinting, plasmid typing and DNA and gene fingerprinting were evaluated. The biochem. fingerprinting system (the PhP-system), discriminated well between different species and had also good discriminatory power within e.g. *S.hemolyticus* and *S.saprophyticus* but *S.epidermidis* were more homogeneous. The DNA fingerprinting, using *BclI*, was an effective epidemiol. marker for *S.epidermidis*. When using a panel of random genomic *S.epidermidis* DNA clones as probes in a Southern blot technique, almost the same level of discrimination was obtained but the results were easier to interpret. The plasmid profile patterns were unstable and did not correlate with the DNA fingerprint pattern. The antibiogram and biotyping discriminated poorly.

L15 ANSWER 9 OF 14 MEDLINE on STN

ACCESSION NUMBER: 92148109 MEDLINE

DOCUMENT NUMBER: PubMed ID: 1686044

TITLE: Outbreak of lethal **coagulase-negative**
staphylococcal infection in a transplant unit: an
epidemiological study.

AUTHOR: Wilton J; Jung K; Nystrom B; Ringden O; **Flock J I**
 SOURCE: Journal of hospital infection, (1991 Dec) 19 (4) 287-9.
 Journal code: 8007166. ISSN: 0195-6701.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Letter
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199203
 ENTRY DATE: Entered STN: 19920405
 Last Updated on STN: 19950206
 Entered Medline: 19920316

L15 ANSWER 10 OF 14 DISSABS COPYRIGHT (C) 2004 ProQuest Information and Learning Company; All Rights Reserved on STN

ACCESSION NUMBER: 87:1473 DISSABS Order Number: AARC018841 (not available for sale by UMI)
 TITLE: THE GENES FOR STAPHYLOCOCCAL PROTEIN A AND STREPTOCOCCAL PROTEIN G
 AUTHOR: **GUSS, BENGT MIKAEL [FIL.DR]**
 CORPORATE SOURCE: UPPSALA UNIVERSITET (SWEDEN) (0903)
 SOURCE: Dissertation Abstracts International, (1987) Vol. 49, No. 1C, p. 53. Order No.: AARC018841 (not available for sale by UMI). ALMQVIST & WIKSELL INTERNATIONAL, STOCKHOLM, SWEDEN. 50 pages.
 ISBN: 91-554-2070-2.
 DOCUMENT TYPE: Dissertation
 FILE SEGMENT: DAI
 LANGUAGE: English
 ENTRY DATE: Entered STN: 19921118
 Last Updated on STN: 19921118

AB The genes coding for protein A from *Staphylococcus aureus* and protein G from a streptococcal strain of serological group G have been isolated by molecular cloning in *Escherichia coli*. The nucleotide sequences of the structural genes and their 5' and 3' flanking regions have been determined.

The deduced nucleotide sequence of the protein A gene reveals a fifth structural region homologous to the four repetitive IgG-binding regions earlier reported. The C-terminal part of protein A, region X, is composed of two structural different domains, an octapeptide structure (Xr) repeated approximately 12 times followed by a region of non-repetitive nature called Xc. The expression of the gene in different hosts was studied by cloning the complete gene and a truncated form, lacking region X, in a shuttle vector. The gene was expressed in *E. coli*, different strains of *S. aureus* and in several species of **coagulase-negative staphylococci**. The protein lacking region X was exclusively extracellular in all *Staphylococcus* species tested, which confirms the importance of region X for cell wall binding.

The deduced amino acid sequence of protein G contains several different repetitive regions. The IgG-binding part of the molecule consists of three highly homologous regions which share no homology with the corresponding regions of protein A. The C-terminal part of protein G has similar structural characteristics as the corresponding region in protein A.

From sequence analysis it is suggested that the repetitive regions in protein A and G, have evolved through a similar stepwise gene duplication mechanism of primordial ancestor fragments generating the repetitive

structures of these genes.

The IgG-binding spectrum of protein G is somewhat different from that of protein A which might imply the use of this protein as a tool in immunological techniques.

L15 ANSWER 11 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 85221542 EMBASE

DOCUMENT NUMBER: 1985221542

TITLE: Virulence of *Staphylococcus aureus* in a mouse mastitis model: Studies of alpha hemolysin, coagulase, and protein A as possible virulence determinants with protoplast fusion and gene cloning.

AUTHOR: Jonsson P.; **Lindberg M.**; Haraldsson I.; Wadstrom T.

CORPORATE SOURCE: National Veterinary Institute, S-750 07 Uppsala, Sweden
SOURCE: Infection and Immunity, (1985) 49/3 (765-769).

CODEN: INFIBR

COUNTRY: United States

DOCUMENT TYPE: Journal

FILE SEGMENT: 004 Microbiology
022 Human Genetics

LANGUAGE: English

AB Mutants of a genetically well-characterized strain of ***Staphylococcus aureus*** [SA113(83A)] were isolated after mutagenization. Alpha-hemolysin- (hla), coagulase- (coa), and protein A- (spa) negative mutants were characterized by more than 90 biochemical tests for production of extracellular proteins and biochemical profile to exclude pleiotropy. Protoplast fusion was then used to isolate double-defective (hla and coa) recombinants and and recombinants with regained properties, i.e., production of alpha-hemolysin and coagulase. Studies of such mutants and recombinants in the mouse mastitis model showed that one alpha-hemolysin [SA113(83A) hla-5] and one **coagulase-negative** [SA113(83A) coa-147] mutant were lower in virulence compared with the wild-type strain SA113(83A). The double-negative mutant SA113(83A) hla-5 coa-147 showed a drastic decline in virulence and only induced very mild changes, as determined by microscopic examinations of infected mammary gland tissue. The recombinant with regained properties, however, was as virulent as the wild-type strain. This suggests that alpha-hemolysin and coagulase are virulence determinants of *S. aureus*. A high-level protein A-producing mutant (U300) showed the same virulence as the parent strain SA113(83A) in this model. One low virulence protein A-negative mutant (U320) did not markedly increase in virulence when a plasmid containing the cloned gene for protein A (pSPA15) was introduced into this mutant. By these and earlier observations, it seems likely that protein A is not an important virulence determinant in mastitis of mice. The reduced virulence of the protein A-negative mutant U320 compared with the wild-type SA113(83A) may be due to pleiotropic loss of some other unknown virulence determinant(s). Our data confirm earlier findings that pleiotropic changes are common in protein A-negative mutants.

L15 ANSWER 12 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1984:449555 CAPLUS

DOCUMENT NUMBER: 101:49555

TITLE: Staphylococcal protein, a coding gene (DNA) fragment

INVENTOR(S): comprising a signal DNA sequence
Loefeldahl, Sven; Uhlen, Mathias; **Lindberg, Martin**
PATENT ASSIGNEE(S): Pharmacia AB, Swed.
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 8400774	A1	19840301	WO 1983-SE298	19830823
W: DE, JP, US				
SE 8204811	A	19840224	SE 1982-4811	19820823
JP 59501694	T2	19841011	JP 1983-502890	19830823
DE 3390106	T	19841115	DE 1983-3390106	19830823
PRIORITY APPLN. INFO.:			SE 1982-4811	19820823
			WO 1983-SE298	19830823

AB DNA sequences that code for the signal peptide of the protein A precursor of *Staphylococcus aureus* and the use of these sequences in the construction of cloning vectors are described. The expression of such signal DNA sequences, when they are joined to cloned genes, allows the secretion of the gene product through a cell membrane. Thus, chromosomal DNA from *S. aureus* strain 8325-4 (.vphi.11) mec-4916, str-4916, nov-142 was cloned in *Escherichia coli* with plasmid pBR322 as the vector. Clones that were ampicillin resistant and tetracycline sensitive were examined for protein A formation by an ELISA procedure. One protein A-producing clone, which was deposited as strain DSM 2434, contained the protein A gene on plasmid pSPA1. A restriction map of pSPA1 was prepared, and the protein A gene was subcloned. The protein A gene and promoter were present on a 2.15-kilobase EcoRV insert. The protein A gene present on subclone pSPA3 was sequenced, and the protein sequence was predicted. The signal peptide of the protein A precursor was expressed in *E. coli* containing plasmid pSPA1, and the protein was exported to the periplasmic space. The plasmids pSPA15 and pSPA16, which could replicate in *E. coli*, *S. aureus*, and **coagulase-neg. staphylococci**, were constructed. Plasmid pSPA15 contained the entire structural gene for protein A. Plasmid pSPA16 encoded a 3'-truncated protein A mol. that contained almost all IgG-binding regions. Both pSPA15 and pSPA16 were used to transform a *S. aureus* protein A- mutant (U320), *S. epidermidis* 247, and *S. xylosus* KL117. The protein A encoded by pSPA15 was bound to cell walls; the protein encoded by pSPA16, which lacked the cell wall-binding region of protein A, was secreted into the medium by the *Staphylococcus* strains.

L15 ANSWER 13 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 1984:545087 CAPLUS
DOCUMENT NUMBER: 101:145087
TITLE: Expression of the gene encoding protein A in
Staphylococcus aureus and **coagulase-negative staphylococci**
AUTHOR(S): Uhlen, Mathias; Guss, Bengt; Nilsson,
Bjoern; Goetz, Friedrich; **Lindberg, Martin**

CORPORATE SOURCE: Dep. Biochem. Biotechnol., R. Inst. Technol.,
Stockholm, S-100 44, Swed.
SOURCE: Journal of Bacteriology (1984), 159(2), 713-19
CODEN: JOBAAY; ISSN: 0021-9193
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Two shuttle vectors containing the gene for protein A (spa) from *S. aureus* were constructed to study expression of the gene in various strains of *S. aureus* and in the coagulase-neg. species *S. epidermidis*, *S. capitis*, and *S. xylosus*. One plasmid, pSPA15, contains the complete structural gene for protein A, which binds to the cell wall in various *Staphylococcus* species. The other plasmid, pSPA16, codes for a truncated protein A that lacks the C-terminal part called region X. The latter is exclusively extracellular in all *Staphylococcus* species examined, which confirms the importance of region X for binding of the cell wall. The expression of the plasmid-encoded protein A in various strains of *S. aureus* is strongly correlated to the expression of the chromosomal spa gene. The coagulase-neg. species expressing plasmid-encoded protein A produce 12-30% of the amount encoded by the chromosomal spa gene in *S. aureus* strains Cowan I and A676.

L15 ANSWER 14 OF 14 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 81117041 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7007333
TITLE: Plasmid transfer and genetic recombination by protoplast fusion in staphylococci.
AUTHOR: Gotz F; Ahrne S; Lindberg M
SOURCE: Journal of bacteriology, (1981 Jan) 145 (1) 74-81.
Journal code: 2985120R. ISSN: 0021-9193.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198104
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810424

AB The experimental conditions for plasmid transfer and genetic recombination in *Staphylococcus aureus* and some coagulase-negative staphylococci by protoplast fusion are described. Protoplasts were prepared by treatment with lysostaphin and lysozyme in a buffered medium with 0.7 to 0.8 M sucrose. Regeneration of cell walls was accomplished on a hypertonic agar medium containing succinate and bovine serum albumin. Transfer of plasmids occurred after treatment of the protoplast mixtures with polyethylene glycol (molecular weight, 6,000) not only between strains of the same species but also between parents of different species, although at approximately 100 times lower frequency in the latter case. Recombination of the chromosomal genes in fused protoplasts required simultaneous treatment of the mixed protoplasts with polyethylene glycol and CaCl₂. A method was developed for isolation of recombinants after fusion between mutants of *S. aureus* carrying unselectable markers. Antibiotic resistance plasmids were introduced into the parental strains and used as primary markers to detect protoplast fusion. Chromosomal recombinants were found among the clones with both parental plasmids at a high frequency. The method appears to have simple applications in the construction of strains with multiple

10/806288

mutant characters.

(FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO, PHIC, PHIN, TOXCENTER, PASCAL, FEDRIP, DISSABS' ENTERED AT 09:22:35 ON 06 OCT 2004)

L16 7 S (L1 OR L2 OR L3 OR L4 OR L5 OR L7 OR L8 OR L9 OR L10)
 AND CNS(S) STAPHYLOCOCC?
L17 0 S L16 NOT L14

FILE 'HOME' ENTERED AT 09:23:47 ON 06 OCT 2004

10/806288

06oct04 08:23:00 User219783 Session D2047.2

SYSTEM:OS - DIALOG OneSearch

File 65:Inside Conferences 1993-2004/Oct W1

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File 440:Current Contents Search(R) 1990-2004/Oct 06

(c) 2004 Inst for Sci Info

File 348:EUROPEAN PATENTS 1978-2004/Sep W04

(c) 2004 European Patent Office

File 357:Derwent Biotech Res. 1982-2004/Oct W1

(c) 2004 Thomson Derwent & ISI

File 113:European R&D Database 1997

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*File 113: This file is closed (no updates)

Set	Items	Description
S1	53	AU=(GUSS, B? OR GUSS B)
S2	1357	AU=(NILSSON, M? OR NILSSON M?)
S3	58	AU=(FRYKBERG, L? OR FRYKBERG L?)
S4	193	AU=(FLOCK, J? OR FLOCK J?)
S5	543	AU=(LINDBERG, M? OR LINDBERG M?)
S6	3	S1 AND S2 AND S3 AND S4 AND S5
S7	39	S1 AND (S2 OR S3 OR S4 OR S5)
S8	15	S2 AND (S3 OR S4 OR S5)
S9	19	S3 AND (S4 OR S5)
S10	6	S4 AND S5
S11	21	(S1 OR S2 OR S3 OR S4 OR S5) AND (((COAGULASE(W) (NEG OR NEGATIV?)) (S) STAPHYLOCOCC?) OR CNS(S) STAPHYLOCOCC?)
S12	14	RD (unique items)

>>>No matching display code(s) found in file(s): 65, 113

- Author(s)

12/3,AB/1 (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)
(c) 2004 Inst for Sci Info. All rts. reserv.

18456683 Document Delivery Available: 000221218300022 References: 28
TITLE: A von Willebrand factor-binding protein from Staphylococcus
lugdunensis

AUTHOR(S): Nilsson M; Bjerketorp J; Wiebensjo A; Ljungh A;

Frykberg L; Guss B (REPRINT)

AUTHOR(S) E-MAIL: bengt.guss@mikrob.slu.se

CORPORATE SOURCE: Swedish Univ Agr Sci, Dept Microbiol, /SE-75007

Uppsala//Sweden/ (REPRINT); Swedish Univ Agr Sci, Dept Microbiol,

/SE-75007 Uppsala//Sweden/; Lund Univ, Dept Med Microbiol Dermatol &

Infect, /SE-22362 Lund//Sweden/

PUBLICATION TYPE: JOURNAL

PUBLICATION: FEMS MICROBIOLOGY LETTERS, 2004, V234, N1 (MAY 1), P155-161

GENUINE ARTICLE#: 818AP

PUBLISHER: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS

ISSN: 0378-1097

LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: In the present study, a phage display library covering the genome of Staphylococcus lugdunensis, was affinity-selected against von Willebrand

factor (vWf). This led to the identification of a gene, vwbl, encoding a Putative cell surface protein of 2060 amino acids, denoted vWbl. The deduced protein has an overall organisation typical of staphylococcal cell Surface proteins, with an N-terminal signal peptide, and a C-terminal cell wall sorting signal. The vWf-binding part is located in repetitive domains and antibodies against vWbl or vWf can inhibit the binding. Southern blot analysis showed that vwbl was present in the 12 *S. lugdunensis* strains tested. (C) 2004 Federation of European Microbiological Societies. Published by Elsevier B.V. All rights reserved.

12/3,AB/2 (Item 2 from file: 440)
 DIALOG(R)File 440:Current Contents Search(R)
 (c) 2004 Inst for Sci Info. All rts. reserv.

16053698 Document Delivery Available: 000182501500002 References: 49
 TITLE: Extracellular adherence protein from *Staphylococcus aureus* enhances internalization into eukaryotic cells

AUTHOR(S): Hagggar A; Hussain M; Lonnie H; Herrmann M; Norrby-Teglund A;

Flock JI (REPRINT)

AUTHOR(S) E-MAIL: jan-ingmar.flock@labmed.ki.se

CORPORATE SOURCE: Huddinge Univ Hosp, Div Clin Bacteriol, F 82/S-14186

Huddinge//Sweden/ (REPRINT); Huddinge Univ Hosp, Div Clin Bacteriol,

/S-14186 Huddinge//Sweden/; Huddinge Univ Hosp, Dept Med, /S-14186

Huddinge//Sweden/; Univ Muenster Hosp, Inst Med Microbiol, /D-48129

Munster//Germany/; Univ Saarland Hosp, Inst Microbiol & Hyg, /D-66421

Homburg//Germany/

PUBLICATION TYPE: JOURNAL

PUBLICATION: INFECTION AND IMMUNITY, 2003, V71, N5 (MAY), P2310-2317

GENUINE ARTICLE#: 672BT

PUBLISHER: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904, USA

ISSN: 0019-9567

LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: In this study we have shown that Eap (extracellular adherence protein) plays a role in the internalization process of *Staphylococcus aureus* into eukaryotic cells. Eap is a protein that is mostly extracellularly and to a lesser extent is bound to the bacterial surface as a result of rebinding. Eap is able to bind to several plasma proteins, such as fibronectin, fibrinogen, and prothrombin. It has the capacity to form oligomers and is able to agglutinate *S. aureus*. A mutant strain, Newman mAH12 (eap:: Ery(r)), with a deficient eap gene was used in the present study. We have demonstrated that (i) strain Newman mAH12 could adhere to and become internalized to a higher extent by eukaryotic cells than the isogenic mutant, (ii) strain Newman mAH12 complemented with the eap gene displayed restoration of the internalization level, (iii) externally added Eap enhanced the internalization of laboratory and clinical *S. aureus* strains as well as of *S. carnosus* (a ~~coagulase-negative~~ species devoid of proteins important for internalization), and (iv) antibodies against Eap were able to block the internalization process in strain Newman mAH12 and clinical isolates. Eap, with its broad binding capacity and its surface localization, thus seems to contribute to the internalization of *S. aureus* into eukaryotic cells. We therefore propose a novel internalization pathway for *S. aureus* in which Eap plays an enhancing role.

12/3,AB/3 (Item 3 from file: 440)
 DIALOG(R)File 440:Current Contents Search(R)
 (c) 2004 Inst for Sci Info. All rts. reserv.

15751599 Document Delivery Available: 000181406500035 References: 19
 TITLE: GST-Fbe can recognize beta-chains of fibrin(ogen) on explanted materials
 AUTHOR(S): Pei L (REPRINT); Arvholm IL; Lonnie L; Flock JI
 AUTHOR(S) E-MAIL: lpei@partners.org
 CORPORATE SOURCE: Massachusetts Gen Hosp, Div Infect Dis, 55 Fruit St,GRJ 504/Boston//MA/02114 (REPRINT); Huddinge Univ Hosp, Div Clin Bacteriol, /SE-14186 Stockholm//Sweden/
 PUBLICATION TYPE: JOURNAL
 PUBLICATION: JOURNAL OF CHROMATOGRAPHY B-ANALYTICAL TECHNOLOGIES IN THE BIOMEDICAL AND LIFE SCIENCES, 2003, V786, N1-2 (MAR 25), P319-325
 GENUINE ARTICLE#: 652XQ
 PUBLISHER: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS
 ISSN: 1570-0232
 LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: Staphylococcus epidermidis, a **coagulase-negative staphylococcus** (CONS), is one of the leading pathogens of nosocomial infections, particularly associated with foreign body infections. Adherence of S. epidermidis to fibrinogen deposited on the surfaces of implants is important for the development of foreign body infections. A gene (fbe) encoding a fibrinogen-binding protein from S. epidermidis (Fbe) was identified by shotgun phage display. A portion of The was cloned into a GST-fusion vector. Affinity to glutathione-Sepharose by the GST-tag and affinity to fibrinogen-Sepharose by the Fbe part were applied to purify the recombinant Fbe. The purity and efficacy of the methods used in protein purification was compared. Furthermore, the potential physiological role of Fbe was studied by the interaction between GST-Fbe and components extracted from explanted materials in vitro. (C) 2002 Elsevier Science B.V. All rights reserved.

12/3,AB/4 (Item 4 from file: 440)
 DIALOG(R)File 440:Current Contents Search(R)
 (c) 2004 Inst for Sci Info. All rts. reserv.

13126263 References: 25
 TITLE: Lack of fbe, the gene for a fibrinogen-binding protein from Staphylococcus epidermidis, reduces its adherence to fibrinogen coated surfaces
 AUTHOR(S): Pei L; Flock JI (REPRINT)
 AUTHOR(S) E-MAIL: jan-ingmar.flock@impi.ki.se
 CORPORATE SOURCE: Karolinska Inst, Div Clin Bacteriol, /S-14186 Stockholm//Sweden/ (REPRINT); Karolinska Inst, Div Clin Bacteriol, /S-14186 Stockholm//Sweden/
 PUBLICATION TYPE: JOURNAL
 PUBLICATION: MICROBIAL PATHOGENESIS, 2001, V31, N4 (OCT), P185-193
 GENUINE ARTICLE#: 479CP
 PUBLISHER: ACADEMIC PRESS LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND
 ISSN: 0882-4010
 LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: The significance of Fbe, a fibrinogen-binding protein in *Staphylococcus epidermidis*, was investigated. A mutant was constructed by allelic replacement, where a Gentamicin resistance gene replaced a portion of the A region of fbe. Adherence assay to immobilized fibrinogen on polyethylene surfaces and peripheral venous catheters from patients showed that the fibrinogen binding ability of the mutant was reduced compared to its parental strain. This shows that Fbe is a major factor involved in adherence of *S. epidermidis* to fibrinogen. No difference was found between the wild-type and mutant in their affinity to immobilized fibronectin. (C) 2001 Academic Press.

12/3,AB/5 (Item 5 from file: 440)
 DIALOG(R)File 440:Current Contents Search(R)
 (c) 2004 Inst for Sci Info. All rts. reserv.

12826351 References: 14

TITLE: Functional study of antibodies against a fibrogenin-binding protein in *Staphylococcus epidermidis* adherence to polyethylene catheters
AUTHOR(S): Pei L; **Flock JI (REPRINT)**
AUTHOR(S) E-MAIL: jan-ingmar.flock@impi.ki.se
CORPORATE SOURCE: Huddinge Univ Hosp F82, Div Clin Bacteriol, /S-14186 Huddinge//Sweden/ (REPRINT); Huddinge Univ Hosp F82, Div Clin Bacteriol, /S-14186 Huddinge//Sweden/
PUBLICATION TYPE: JOURNAL
PUBLICATION: JOURNAL OF INFECTIOUS DISEASES, 2001, V184, N1 (JUL 1), P52-55
GENUINE ARTICLE#: 447DL
PUBLISHER: UNIV CHICAGO PRESS, 1427 E 60TH ST, CHICAGO, IL 60637-2954 USA
ISSN: 0022-1899
LANGUAGE: English **DOCUMENT TYPE:** ARTICLE

ABSTRACT: *Staphylococcus epidermidis* is an important pathogen in foreign body-associated infections. In a previous study, we showed that a surface-located fibrinogen-binding protein, termed Fbe, from *S. epidermidis* mediated the bacterial adherence to fibrinogen-coated surfaces in vitro. In the present study, we demonstrate that antibodies against Fbe can block adherence of *S. epidermidis* to fibrinogen-coated catheters, subcutaneously implanted catheters from rats, and peripheral venous catheters from human patients.

12/3,AB/6 (Item 6 from file: 440)
 DIALOG(R)File 440:Current Contents Search(R)
 (c) 2004 Inst for Sci Info. All rts. reserv.

10843952 References: 36

TITLE: Functional studies of a fibrinogen binding protein from *Staphylococcus epidermidis*
AUTHOR(S): Pei L; Palma M; Nilsson M; Guss B; **Flock JI (REPRINT)**
AUTHOR(S) E-MAIL: jan-ingmar.flock@impi.ki.se
CORPORATE SOURCE: Huddinge Univ Hosp, Dept Immunol Microbiol Pathol & Infect Dis, F82/S-14186 Huddinge//Sweden/ (REPRINT); Huddinge Univ Hosp, Dept Immunol Microbiol Pathol & Infect Dis, /S-14186 Huddinge//Sweden/; Swedish Univ Agr Sci, Dept Microbiol, /S-75007 Uppsala//Sweden/

PUBLICATION TYPE: JOURNAL
 PUBLICATION: INFECTION AND IMMUNITY, 1999, V67, N9 (SEP), P4525-4530
 GENUINE ARTICLE#: 228LU
 PUBLISHER: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW,
 WASHINGTON, DC 20005-4171 USA
 ISSN: 0019-9567
 LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: A gene encoding a fibrinogen binding protein from *Staphylococcus epidermidis* was previously cloned, and the nucleotide sequence was determined. A portion of the gene encompassing the fibrinogen binding domain has now been subcloned in an expression-fusion vector. The fusion protein can bind to fibrinogen in a capture enzyme-linked immunosorbent assay and can be purified by fibrinogen affinity chromatography. This protein can completely inhibit the adherence of *S. epidermidis* to immobilized fibrinogen, suggesting that the adherence of *S. epidermidis* to fibrinogen is mainly due to this protein. Antibodies against this fibrinogen binding protein were also found to efficiently block the adherence of *S. epidermidis* to immobilized fibrinogen. Despite homology with clumping factors A and B from *S. aureus* (cell surface-associated proteins binding to fibrinogen), binding involved the beta chain of fibrinogen rather than the gamma chain, as in clumping factor A.

12/3,AB/7 (Item 7 from file: 440)
 DIALOG(R)File 440:Current Contents Search(R)
 (c) 2004 Inst for Sci Info. All rts. reserv.

09500860 References: 38
 TITLE: A fibrinogen-binding protein of *Staphylococcus epidermidis*
 AUTHOR(S): Nilsson M; Frykberg L; Flock JI; Pei L;
 Lindberg M; Guss B (REPRINT)
 CORPORATE SOURCE: SWEDISH UNIV AGR SCI,DEPT MICROBIOL, BOX 7025/S-75007
 UPPSALA//SWEDEN/ (REPRINT); SWEDISH UNIV AGR SCI,DEPT MICROBIOL/S-75007
 UPPSALA//SWEDEN/; HUDDINGE UNIV HOSP,DEPT IMMUNOL MICROBIOL PATHOL &
 INFECT DIS, KAROLINSKA INST/S-14186 HUDDINGE//SWEDEN/
 PUBLICATION TYPE: JOURNAL
 PUBLICATION: INFECTION AND IMMUNITY, 1998, V66, N6 (JUN), P2666-2673
 GENUINE ARTICLE#: ZP714
 PUBLISHER: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW,
 WASHINGTON, DC 20005-4171
 ISSN: 0019-9567
 LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: The present study reports on fibrinogen (Fg) binding of *Staphylococcus epidermidis*. Adhesion of different *S. epidermidis* strains to immobilized Fg was found to vary significantly between different strains, and the component responsible was found to be proteinaceous in nature. To further characterize the Fg-binding activity, a shotgun phage display library covering the *S. epidermidis* chromosome was constructed. By affinity selection (panning) against immobilized Fg, a phagemid clone, pSEFG1, was isolated, which harbors an insert with an open reading frame of similar to 1.7 kilobases. Results from binding and inhibition experiments demonstrated that the insert of pSEFG1 encodes a specific Fg-binding protein. Furthermore, affinity-purified protein encoded by pSEFG1 completely inhibited adhesion of *S. epidermidis* to immobilized Fg. By additional

cloning and DNA sequence analyses, the complete gene, termed fbe, was found to consist of an open reading frame of 3,276 nucleotides encoding a protein, called Fbe, with a deduced molecular mass of similar to 119 kDa. With a second phage display library made from another clinical isolate of *S. epidermidis*, it was possible to localize the Fg-binding region to a 331-amino-acid-long fragment. PCR analysis showed that the fbe gene was found in 40 of 43 clinical isolates of *S. epidermidis*. The overall organization of Fbe resembles those of other extracellular surface proteins of staphylococci and streptococci. Sequence comparisons with earlier known proteins revealed that this protein is related to an Fg-binding protein of *Staphylococcus aureus* called clumping factor.

12/3,AB/8 (Item 8 from file: 440)
 DIALOG(R)File 440:Current Contents Search(R)
 (c) 2004 Inst for Sci Info. All rts. reserv.

06894540 References: 20

TITLE: TYPING OF COAGULASE-NEGATIVE **STAPHYLOCOCCI** FROM PERITONITIS IN CAPD PATIENTS BY THE PHP-CS SYSTEM AND REA

AUTHOR(S): JUNG K; BRAUNER A; KUHN I; RANSJO U; HYLANDER B; **FLOCK JI**; MOLLBY R

CORPORATE SOURCE: DANDERYD HOSP,DEPT CLIN BACTERIOL/S-18288

DANDERYD//SWEDEN/ (Reprint); KAROLINSKA HOSP,CTR MICROBIOL & TUMORBIOL/S-10401 STOCKHOLM//SWEDEN//; KAROLINSKA HOSP,DEPT CLIN MICROBIOL/S-10401 STOCKHOLM//SWEDEN//; KAROLINSKA HOSP,DEPT MED,DIV NEPHROL/S-10401 STOCKHOLM//SWEDEN//; KAROLINSKA INST,NOVUM,CTR BIOTECHNOL/HUDDINGE//SWEDEN/

PUBLICATION: APMIS, 1995, V103, N9 (SEP), P679-685

GENUINE ARTICLE#: TE188

ISSN: 0903-4641

LANGUAGE: ENGLISH DOCUMENT TYPE: ARTICLE

ABSTRACT: Coagulase-negative **staphylococci** (**CNS**) were the most common bacteria causing peritonitis in patients treated with continuous ambulatory peritoneal dialysis (CAPD). In order to investigate if the same clone was responsible for the peritonitis in the different patients and if the exit site was the source of infection we followed 68 patients on CAPD for 2 years. During this period 9 patients had 12 episodes of peritonitis caused by **CNS**. Cultures were taken from exit site and peritoneal fluid in all patients at peritonitis and during the first study year at monthly intervals. In each culture up to 10 isolates of **CNS** were randomly collected and frozen. All 437 **CNS** isolates from the patients with **CNS** peritonitis were typed using a biochemical typing method and 41 isolates identical by this method were further discriminated by a DNA fingerprinting method. Identical strains were in no case isolated from different patients, indicating that no virulent strain was spread between the patients. The isolates causing the peritonitis were never found at the exit sites before the first day of the peritonitis in any patient. In only two patients was the same strain found at the exit site and in the peritoneal fluid on the first day of peritonitis. It thus seems that no virulent clone of **CNS** was infecting the patients and we found no evidence of **CNS** at the exit site causing the peritonitis.

12/3,AB/9 (Item 9 from file: 440)

DIALOG(R)File 440:Current Contents Search(R)
(c) 2004 Inst for Sci Info. All rts. reserv.

03913063 References: 26

TITLE: COMPARATIVE EVALUATION OF A NEW MOLECULAR METHOD FOR TYPING
STAPHYLOCOCCUS-EPIDERMIDIS

AUTHOR(S): WILTON J; JUNG K; VEDIN I; ARONSSON B; **FLOCK JI**

CORPORATE SOURCE: KAROLINSKA INST,CTR BIOTECHNOL/S-14152 HUDDINGE//SWEDEN/
(Reprint); STOCKHOLM CTY COUNCIL,CENT MICROBIOL LAB/S-10726
STOCKHOLM//SWEDEN//; CLIN RES CTR,MICROBIAL PATHOGENIC RES GRP/HARROW HA1
3UJ/MIDDY/ENGLAND/

PUBLICATION: EUROPEAN JOURNAL OF CLINICAL MICROBIOLOGY & INFECTIOUS
DISEASES, 1992, V11, N6 (JUN), P515-521

GENUINE ARTICLE#: JJ179

LANGUAGE: ENGLISH DOCUMENT TYPE: ARTICLE

ABSTRACT: The discriminatory powers of several techniques for typing Staphylococcus epidermidis were evaluated in an epidemiological study of bacteria isolated from intensive care patients and from neonates. Genomic DNA fingerprinting using BclI restriction endonuclease was an effective epidemiological marker. The distinct restriction fragment profiles produced with this enzyme were highlighted with specific probes in a Southern blot technique. Cloned Escherichia coli rRNA gene probes proved to have lower discriminatory power and be less suitable for intraspecies typing. However, a panel of random genomic Staphylococcus epidermidis DNA clones provided almost the same level of discrimination as the DNA fingerprinting technique and also provided a clearer profile. DNA and gene fingerprinting techniques were reproducible and highly discriminatory compared to typing based on antigen and plasmid profiles, antibiotic susceptibility patterns and biotypes.

12/3,AB/10 (Item 10 from file: 440)

DIALOG(R)File 440:Current Contents Search(R)
(c) 2004 Inst for Sci Info. All rts. reserv.

03379507 References: 5

TITLE: OUTBREAK OF LETHAL COAGULASE-NEGATIVE STAPHYLOCOCCAL INFECTION
IN A TRANSPLANT UNIT - AN EPIDEMIOLOGICAL STUDY

AUTHOR(S): WILTON J; JUNG K; NYSTROM B; RINGDEN O; **FLOCK JI**

CORPORATE SOURCE: KAROLINSKA INST,CTR BIOTECHNOL/S-14152 HUDDINGE//SWEDEN/
(Reprint); STOCKHOLM CTY COUNCIL,CENT MICROBIOL LAB/S-10726
STOCKHOLM//SWEDEN//; HUDDINGE HOSP,DEPT TRANSPLANT SURG/S-14186
HUDDINGE//SWEDEN//; HUDDINGE HOSP,DEPT CLIN MICROBIOL/S-14186
HUDDINGE//SWEDEN//; HUDDINGE HOSP,DEPT CLIN IMMUNOL/S-14186
HUDDINGE//SWEDEN/

PUBLICATION: JOURNAL OF HOSPITAL INFECTION, 1991, V19, N4 (DEC), P287-289

GENUINE ARTICLE#: GY839

LANGUAGE: ENGLISH DOCUMENT TYPE: LETTER

12/3,AB/11 (Item 1 from file: 348)

DIALOG(R)File 348:EUROPEAN PATENTS
(c) 2004 European Patent Office. All rts. reserv.

00917082

NEW FIBRINOGEN BINDING PROTEIN ORIGINATING FROM COAGULASE-
NEGATIVE STAPHYLOCOCCUS
 NEUARTIGES, FIBRINOGEN-BINDENDES PROTEIN, WELCHES VON COAGULASE-
NEGATIVEN STAPHYLOCOCCUS ABSTAMMT
 NOUVELLE PROTEINE DE FIXATION DE FIBRINOGENE TIRANT SON ORIGINE DU
 STAPHYLOCOQUE COAGULASE-NEGATIF

PATENT ASSIGNEE:

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 AT;BE;CH;DE;DK;ES;FI;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
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 AT;BE;CH;DE;DK;ES;FI;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
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 AT;BE;CH;DE;DK;ES;FI;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)
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 designated states:
 AT;BE;CH;DE;DK;ES;FI;FR;GB;GR;IE;IT;LI;LU;MC;NL;PT;SE)

INVENTOR:

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 Flock, Jan-Ingmar, Sangarvagen 2, 161 28 Bromma, (SE)
 Lindberg, Martin, Sveriges Lantbruksuniversitet, Inst. for
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LEGAL REPRESENTATIVE:

Onn, Thorsten et al (23893), AB STOCKHOLMS PATENTBYRA Box 23101, 104 35
 Stockholm, (SE)

PATENT (CC, No, Kind, Date): EP 922056 A1 990616 (Basic)

WO 9748727 971224

APPLICATION (CC, No, Date): EP 97928617 970618; WO 97SE1091 970618

PRIORITY (CC, No, Date): SE 962496 960620

DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FI; FR; GB; GR; IE; IT; LI; LU;
 MC; NL; PT; SE

INTERNATIONAL PATENT CLASS: C07K-014/31;

NOTE:

No A-document published by EPO

LANGUAGE (Publication,Procedural,Application): English; English; English

12/3,AB/12 (Item 2 from file: 348)

DIALOG(R)File 348:EUROPEAN PATENTS

(c) 2004 European Patent Office. All rts. reserv.

00619809

FIBRINOGEN BINDING PROTEIN

FIBRINOGEN-BILDUNGSPROTEIN

PROTEINE DE LIAISON DU FIBRINOGENE

PATENT ASSIGNEE:

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FLOCK, Jan-Ingmar, Sangarvagen 2, S-161 28 Bromma, (SE)

LEGAL REPRESENTATIVE:

Bergvall-Eftring, Stina-Lena et al (22401), Dr. Ludwig Brann Patentbyra

AB P.O. Box 17192, 104 62 Stockholm, (SE)

PATENT (CC, No, Kind, Date): EP 621875 A1 941102 (Basic)

EP 621875 B1 020327

WO 9406830 940331

APPLICATION (CC, No, Date): EP 93921159 930920; WO 93SE759 930920

PRIORITY (CC, No, Date): SE 922720 920921; SE 932955 930913

DESIGNATED STATES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LI; LU; MC;

NL; PT; SE

INTERNATIONAL PATENT CLASS: A61K-039/085; C12N-009/48

NOTE:

No A-document published by EPO

LANGUAGE (Publication, Procedural, Application): English; English; English

FULLTEXT AVAILABILITY:

Available Text	Language	Update	Word Count
CLAIMS B	(English)	200213	167
CLAIMS B	(German)	200213	141
CLAIMS B	(French)	200213	188
SPEC B	(English)	200213	7590
Total word count - document A			0
Total word count - document B			8086
Total word count - documents A + B			8086

12/3,AB/13 (Item 1 from file: 357)

DIALOG(R) File 357: Derwent Biotech Res.

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0221634 DBR Accession No.: 98-03231 PATENT

New fibrinogen binding protein from **coagulase-negative**

Staphylococcus - Staphylococcus epidermidis recombinant protein expression using a plasmid, phage or phagemid vector, for use as a recombinant vaccine or nucleic acid vaccine

AUTHOR: Guss B; Nilsson M; Frykberg L; Flock J I
; Lindberg M

CORPORATE SOURCE: Uppsala, Sweden; Bromma, Sweden.

PATENT ASSIGNEE: Guss B; Nilsson M; Frykberg L; Flock J I; Lindberg M 1997

PATENT NUMBER: WO 9748727 PATENT DATE: 971224 WPI ACCESSION NO.: 98-063079 (9806)

PRIORITY APPLIC. NO.: SE 962496 APPLIC. DATE: 960620

NATIONAL APPLIC. NO.: WO 97SE1091 APPLIC. DATE: 970618

LANGUAGE: English

ABSTRACT: A new fibrinogen binding protein is isolated from a **coagulase-negative Staphylococcus** sp. DNA encoding the protein may be inserted in a plasmid, phage or phagemid vector, for expression in a microorganism. The resulting recombinant protein may be purified from a culture of the microorganism by chromatography. An antibody specific for the protein is also new, and may be used in diagnostic, therapeutic or prophylactic applications, or to block adherence of **staphylococci**. The protein or DNA may be used as a recombinant vaccine or a nucleic acid vaccine, respectively, and the

antibody may be used in passive immunization. A DNA probe based on the sequence may also be used to identify **Staphylococcus** epidermidis, in DNA fingerprinting of strains, and in isolating similar genes from other species. (45pp)

12/3,AB/14 (Item 2 from file: 357)
 DIALOG(R)File 357:Derwent Biotech Res.
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0026652 DBR Accession Number: 84-09927

Expression of the gene encoding protein A in **Staphylococcus aureus** and **coagulase-negative staphylococci** - construction and application of 2 shuttle vectors

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JOURNAL: J.Bacteriol. (159, 2, 713-19) 1984

CODEN: JOBAAY

LANGUAGE: English

ABSTRACT: 2 Shuttle vectors containing the gene for protein A (spa) from **Staphylococcus aureus** have been constructed to analyze expression of the gene in Staph. aureus strains and in the **coagulase-negative species Staphylococcus epidermidis, Staphylococcus capitis** and **Staphylococcus xylosus**. Plasmid pHV33 was used for cloning. pSPA3 was used to supply the spa gene which was ligated into pHV33. The isolated plasmid, pSPA15, contained the complete structural gene for protein A. Plasmid pSPA16 was constructed and contained a deleted spa gene coding for a truncated protein A. For transfer of plasmids from *Escherichia coli* to **staphylococci**, protoplasts of the mutant Staph. aureus SA113 were used. The expression of plasmid-encoded protein A in the Staph. aureus strains was correlated to expression of the chromosomal spa gene. (45 ref)

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